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THE Rh FACTOR IN BLOOD AND RELATED CONDITIONS*

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The purpose of this paper is to summarize briefly the history and present knowledge of the Rh factor in blood and its practical applications; to correlate these with actual observation. The bibliography is meant to serve as a guide to those who wish to investigate the Rh factor more thoroughly and in greater detail.

The first hint of a new "antigen-antibody" reaction which might be a causative factor in hemolytic anemia of the newborn, was mentioned by Darrow in 1938.[1] However, she presented no evidence to substantiate her view.*

In 1939, Levine and Stetson discovered an agglutinin in the blood of a patient who had delivered a macerated fetus, which agglutinated the cells of most Group O donors. The agglutinin, they believed, was due to isoimmunization. It gradually decreased in titre and in several months disappeared. This indicated an immune body rather than a natural one.[2]

In 1940, while experimenting with rabbit immune sera, Landsteiner and Wiener discovered that serum obtained by injecting the blood of the Rhesus monkey (Macacus) into a rabbit, could be used

*Award paper (First), June, 1945.

to detect a hitherto unknown factor in human blood. This factor, which they designated Rh, agglutinated the majority of human red blood cells, regardless of their blood group, M, N, or P type.[3]

The same year, Wiener and Peters reported three cases of severe hemolytic transfusion reactions to supposedly compatible blood. In two patients, the anti-Rh isoagglutinins were demonstrated in the serum. They assumed the agglutinins to have been stimulated by the transfusion of Rh positive blood into Rh negative individuals.[4]

The Rh Factor in Blood and Related Conditions

Levine and Katzin also reported several cases in which pregnant women experienced severe hemolytic transfusion reactions. The sera of six of these women had demonstrable atypical warm agglutinins which corresponded in specificity to the "anti-Rh" of Lansteiner and Wiener.[5]

Burnham, in 1941, recognized and associated the common cause of erythroblastosis fetalis and hemolytic transfusion reactions.[6]

In the same year, Lansteiner and Wiener described a practical method for testing for the Rh factor and reported an investigation of its heredity.[7] Out of 448 persons, 84.6% were Rh positive and 15.4% were Rh negative. Further investigations brought the following conclusions: The Rh factor is inherited as a simple Mendelian dominant, not sex-linked.

The nomenclature most widely accepted at the present time, is that proposed by Wiener in June, 1944:[8]

Anti-Rh_0'—agglutinin giving 87% positive reactions in a random white population;

Anti-Rh_0—agglutinin giving 85% positive reactions in a random white population;

Anti-Rh′—agglutinin giving 70% positive reactions in a random white population;

Anti-Rh″—agglutinin giving 30% positive reactions in a random white population;

Anti-Rh_0 will react with the agglutinogens Rh_0, Rh_1, Rh_2, Rh_1Rh_2;

Anti-Rh′ will react with the agglutinogens Rh_1, Rh_1Rh_2;

Anti-Rh″ will react with the agglutinogens Rh_2, Rh_1Rh_2.

The Rh type of the offspring is dependent upon the genes inherited from the parents. The following table will serve to illustrate.

Bear in mind that this table indicates only possibilities and not actual ratios.

The Rh Factor in Blood and Related Conditions

MOTHER	OFFSPRING				FATHER
Rh+(RhRh)	Rh+ (RhRh)	Rh+ (RhRh)	Rh+ (RhRh)	Rh+ (RhRh)	Rh+(RhRh)
Rh—(rhrh)	Rh— (rhrh)	Rh— (rhrh)	Rh— (rhrh)	Rh— (rhrh)	Rh—(rhrh)
Rh—(rhrh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh+(RhRh)
Rh+(RhRh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh—(rhrh)
Rh+(Rhrh)	Rh+ (RhRh)	Rh+ (RhRh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh+(RhRh)
Rh+(Rhrh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh+ (RhRh)	Rh— (rhrh)	Rh+(Rhrh)
Rh—(rhrh)	Rh+ (Rhrh)	Rh+ (Rhrh)	Rh— (rhrh)	Rh— (rhrh)	Rh+(Rhrh)

Disregarding sub-types and using only the general classification Rh positive and Rh negative, the following racial distribution has been reported by Wiener, Levine, et al.[9, 10, 13, 17, 21]

	Rh positive	Rh negative
White population	87.25%	12.75%
Negro	93.5 %	6.5 %
Chinese	99.3 %	0.7 %
American Indian	99.2 %	0.8 %

The most satisfactory method for detecting Rh positive and Rh negative blood is dependent upon a potent anti-Rh_0 serum. This serum may be purchased or it may be prepared in one's own laboratory.

Preparing one's own serum necessitates a mother who has recently given birth to an erythroblastotic infant and in whose serum the anti-Rh_0 agglutinin can be demonstrated. This must be in a sufficiently high titre to insure reliability of reactions.

Such a serum is quite rare. More often than not, such a mother demonstrates no anti-Rh agglutinin in her serum although her baby may have a severe case of erythroblastosis fetalis. The explanation for this seems to be in the attachment of the anti-Rh

agglutinin to the tissue cells of the reticulo-endothelial system.[11, 12]

Having found a serum in which the anti-Rh agglutinin can be demonstrated, it is advisable to determine its titre.

Serial dilutions are made of the serum to be titrated as follows: in the first tube, place 0.5 cc. undiluted serum and in each succeeding tube, 0.5 cc. of normal saline solution. To the second tube, add 0.5 cc. serum, mix saline and serum, transfer 0.5 cc. of this mixture to the following tube. Repeat the procedure for each of the subsequent tubes and discard 0.5 cc. diluted serum from the last tube. Each tube now contains 0.5 cc. of progressively doubled dilutions of serum as 1:1; 1:2; 1:4; 1:8, etc.

Set up as many rows of small test tubes (10x75 mm.) as you have different, known Group O Rh positive cells. Carry out the following procedure for each suspension of cells against which unknown serum is to be titrated. (Make 2% saline suspension of washed cells.) Place 0.05 cc. of each dilution of serum in properly labeled tubes. To each tube in the first row, add 0.05 cc. of the first known cell suspension. To each tube in the second row, add 0.05 cc. of the second known cell suspension. Repeat with each cell suspension to be used. Incubate these mixtures in a water bath at 37° C. for one hour. Remove from water bath and centrifuge one minute at 500 rpm. Resuspend the cells by gentle shaking and examine microscopically for agglutination. The dilution of the serum in the last tube in which agglutination is demonstrated, determines the titre of the serum.

The isoagglutinins anti-A and anti-B must then be absorbed to exclude any interference by them in subsequent tests. This is readily accomplished by the addition of Witebsky's group specific substances A and B[24] or by absorption with saliva.

The Rh Factor in Blood and Related Conditions

Diamond has recently suggested a rapid slide test for demonstrating the anti-Rh agglutinin which he considers more accurate than the test tube method.[14] When the anti-Rh agglutinin can not be demonstrated by the test tube method and all clinical evidence suggests that it should be present, he feels that an "inhibiting substance" prevents the reaction from taking place. He consequently presented the following technique. 0.2 cc. of fresh oxalated Rh negative and Rh positive Group O whole blood or washed cells are placed upon an ordinary slide that has been ringed. If washed cells

are used, the suspension is made up to approximately 40-50%. 0.1 cc. of serum to be tested is added to the Rh positive and 0.1 cc. to the Rh negative cells. (The latter acts as a control.) The mixture is gently rotated. The reactions are accelerated at 37° C. but are easily performed at room temperature. In serum from an immunized individual, agglutination is visible within one and three minutes.

Diamond contends that in most sera two factors are concerned, an agglutinin and an inhibitor substance, both capable of attachment to cells. When the inhibiting substance is nearly equivalent, or of stronger titre than the agglutinin, most of the cells in a thin suspension are bound by the inhibiting substance, leaving so few cells to be bound by the agglutinin that visible clumps can not be formed. In heavier suspensions, there are enough cells to absorb the inhibitor substance and to react with the agglutinin in sufficient numbers to form easily visible clumps.

Using this prepared potent serum, the following procedure detects the Rh agglutinogen in the unknown cells. 0.05 cc. anti-Rh_0 serum and 0.05 cc. of a 2% saline suspension of unknown washed cells are placed in a small test tube (10x75 mm.) and incubated in a water bath at 37° C. for one hour. At the end of this time they are centrifuged one minute at 500 rpm. Resuspend cells by gentle shaking and examine macroscopically for agglutination. Should there be any doubt of the reaction, always check the reading microscopically.

A modified compatibility test for those who do not have a potent anti-Rh_0 serum available has been suggested. Two drops of a 2% suspension of patient's cells are mixed with two drops of the donor's serum in a small test tube; likewise the same amounts of patient's serum and donor's cells are mixed in another tube. To each tube, two drops of normal saline are added. The tubes are incubated for thirty to sixty minutes in a water bath at 37° C., centrifuged for one minute at 500 rpm. and read microscopically. This test is done routinely in our laboratory for all cross-matchings. In 5,948 cross-matchings set up by this method, only once has an Rh incompatibility been demonstrated. Consequently, too much reliability must not be placed in this test.

Eleven cases of erythroblastosis fetalis have been observed and studied in this laboratory during the past eighteen months. Although

the anti-Rh agglutinin was definitely established as the causative factor, the sera of only three mothers could be used for diagnostic purposes.

The Rh factor and its accompanying phenomenon of isoimmunization, has its practical application in the syndrome known as erythroblastosis fetalis and in the role it plays in hemolytic transfusion reactions.

Erythroblastosis Fetalis

Erythroblastosis fetalis is a syndrome of the newborn.[15] Its pathogenesis depends upon the isoimmunization of the mother by blood factors in the fetus and subsequent passage of maternal immune agglutinins through the placenta. Intrauterine hemolysis of fetal blood results with a compensatory extra-medullary hematopoiesis. It may be characterized by one or all of the following manifestations, depending upon the severity of the disease.

1. Icterus gravis neonatorum: this is, in part, a hemolytic icterus due to red blood cell destruction, in part, to intrahepatic biliary obstruction and obstructive jaundice.
2. Fetal hydrops: liver cells are damaged, causing impaired liver function which may produce a hypoproteinemia. Hypoproteinemia, in turn, leads to edema.
3. Anemic anoxia: the blood of these infants is characterized by a macrocytosis and usually the presence of many erythroblasts. Erythroblasts, as well as macrocytes, reduce the oxygen-carrying capacity of blood.

Certain very definite conditions must be fulfilled before erythroblastosis fetalis will be produced:

1. the father must be Rh positive;
2. the fetus must be Rh positive;
3. fetal red blood cells must pass the placental barrier;
4. the mother's body must be able to react to the fetal cells with antibody formation;
5. anti-Rh isoantibodies must pass the placental barrier into fetal blood stream.

The following examples, which have come under our observa-

tion, bear out the necessity for fulfilling these conditions.

Case 1 (15636-P). This case is presented to emphasize the fact that fetal cells must pass the placental barrier, immunize the mother, and the returning isoantibodies must pass into the fetal blood stream. In other words, this presupposes an accident to the circulation of the placental villi. It also would refute the theory that once a mother gives birth to an infant with erythroblastosis fetalis, all subsequent pregnancies with infants that are Rh positive, will produce erythroblastotic infants.

A mother, age 39, gave birth to her sixth baby on 12-24-44. The baby, birth weight 6 lbs. 4 oz., was slightly jaundiced. The following day the jaundice was markedly increased. The blood count (12-25-44): Hb. 13 gms./100 cc., rbc 3.93, wbc 15,000; rbc definitely macrocytic with 3 erythroblasts/100 wbc. The spleen and liver were palpable. The mother's blood was found to be Group O —Rh negative: anti-Rh titre 1:2; father, Group A—Rh positive; infant, Group O—Rh positive. The blood count on 12-27-44: Hb. 12 gms./100 cc., rbc 3.1, wbc 10,600; no erythroblasts seen. The baby up until this time had been breast-fed. This was discontinued. (Witebsky has found the anti-Rh agglutinin in breast milk.[16]) On 12-27-44, 40 cc. Group O—Rh negative blood was given, followed by 40 cc. more on 12-28-44. Count: (12-28-44) Hb. 13.5 gms./100 cc., rbc 4.34. By 12-30-44 the jaundice was rapidly decreasing and the baby was discharged on 1-1-45. In March, 1945, the baby was reported in good health.

The other children of the same family were typed with the following results:

♀ child, 19 years old—Group O—Rh positive—normal at birth.

♂ child, 13 years old—Group O—Rh positive—normal at birth.

♂ child, 9 years old—Group A—Rh positive—this child was very jaundiced at birth—spontaneous recovery.
Two years later—Miscarriage—Rh immunization?

♀ child, 5 years old—Group O—Rh positive—perfectly normal at birth.

♀ child, 1 year old—Group O—Rh positive—perfectly normal at birth.

♂ child, present case—Group O—Rh positive—erythroblastosis fetalis.

In spite of the birth of one erythroblastotic infant and a later miscarriage, due probably to isoimmunization to the Rh factor, this mother gave birth uneventfully to two perfectly normal Rh positive babies, following her immunization.

Case 2 (3867-P). A mother, age 26, gave birth to her third living child of five pregnancies. There have been two miscarriages. The baby, weighing 7 lbs. 6 oz., was born on March 31, 1944. Jaundice was noticed which increased in intensity. Count: (4-1-44) Hb. 13.5 gms./100 cc., rbc 3.43, wbc 21,550. Six erythroblasts/100 wbc were seen in the blood smear. Macrocytes and polychromatophilia were marked. The baby's spleen and liver were enlarged and edema was marked. The mother proved to be Group A—Rh negative; anti-Rh titre 1:28. The father and infant were Group O —Rh positive. On 4-7-44, 60 cc. Group O—Rh negative blood was given intravenously, followed by 75 cc. on 4-10-44. On the same day, the baby was discharged from the hospital with a Hb. 13.5 gms./100 cc., rbc 4.3. We were unable to obtain the blood groups of the other two living children.

Case 3 (12481-P). At the age of 36, a mother gave birth to her third baby weighing 9 lbs. 10 oz., on October 7, 1944. The infant appeared jaundiced and edematous. The liver and spleen were markedly enlarged. The blood count: Hb 11.5 gms./100 cc., rbc 3.4, wbc 41,410; smear showed 60 erythroblasts/100 wbc. A transfusion of 60 cc. Group O—Rh negative blood was given into the bone marrow of the tibia. The following day (10-8-44) the baby appeared more deeply jaundiced. A blood count was taken at 1:00 p. m.: Hb. 10 gms./100 cc., rbc 2.52. At 2:15 p. m. 60 cc. Group O—Rh negative blood was given into the tibia. At 5:30 p. m. oxygen therapy was started. At 10:00 p. m. respirations became labored and shallow. By 10:30 p. m. there were long periods of apnea. Dark brown mucus exuded from the nose and mouth. The aspirator and resuscitator were used but respirations ceased at 11:25 p. m.

The mother's blood was Group O—Rh negative with an anti-Rh titre of 1:32. The father was Group O—Rh positive. Two living children, both normal at birth, were tested:

♂ child age 12—Group O—Rh positive
♂ child age 3—Group O—Rh positive

The mother's serum was tested three months after delivery and

the anti-Rh agglutinin was demonstrable in a titre of 1:2 as compared with 1:32 immediately after delivery.

Case 4 (6628-P). A mother gave birth to her second child on March 14, 1943. The baby appeared markedly jaundiced with evidence of petechial hemorrhages. Respirations were irregular and oxygen therapy was started immediately. The count: (3-14-43) Hb. 10 gms./100 cc., rbc 3.31, wbc 58,950. There were 90 erythroblasts/100 wbc in the smear. An x-ray of the chest demonstrated an enlarged thymus. A blood smear on 3-16-43 showed 40 erythroblasts/100 wbc. On 3-22-43, the count was Hb. 5.5 gms./100 cc., rbc 1.56, wbc 27,800 with 26 erythroblasts/100 wbc. On 3-25-43, Hb. 5.0 gms./100 cc., rbc 1.76, 12 erythroblasts/100 wbc. On 3-28-43, 60 cc. Group A, Rh negative blood was given into a scalp vein. On 3-30-43, Hb. 10.5 gms., rbc 3.71, wbc 17,000, 6 erythroblasts/100 wbc. At this point the baby had a very mild jaundice. On 4-5-43, the count was: Hb. 10.5 gms./100 cc., rbc 3.65. The baby was discharged from the hospital on April 13, 1943.

Mother—Group A—Rh negative; anti-Rh titre 1:20.
Father—Group O—Rh positive.
♀ Child—age 4—Group O—Rh positive.

The baby now two years old, is a plump, healthy, vivacious, normal child.

The following table lists six cases in which the mother is Rh negative, the father, Rh positive and two or more normal offspring have been delivered.

Mother Rh— Father Rh+	++ 1st child	2nd child	3rd child	4th child	5th child	6th child
O.P. (W.W.)	normal Rh?	normal Rh—	normal Rh—	normal Rh—	normal Rh+	normal Rh+
O.P. (J.B.)	normal Rh+	normal Rh+	normal Rh+	normal Rh—	jaundiced Rh+	
12481-P	normal Rh+	normal Rh—				
O.P. (E.L.)	normal Rh—	normal Rh—				
14314-P	normal Rh—	normal Rh—	normal Rh—	normal Rh—		
1006-Q	normal Rh+	normal Rh+	normal Rh+	normal Rh—	normal Rh+	

Erythroblastosis Fetalis With Rh Positive Mothers

The Rh factor in blood explains only about 90% of the known cases of erythroblastosis fetalis. About 10% of these cases have

Rh positive mothers.

An atypical agglutinin produced by an Rh mother of an erythroblastotic infant was discovered by Levine.[18, 22] The immune agglutinin reacted on all Rh negative and Rh_2 bloods, hence, it is thought to bear a genetic relationship to the Rh factor. For this reason, this agglutinin was termed Hr (Rh reversed) and its corresponding antibody, anti-Hr.

Race and Taylor observed an agglutinin,[19] apparently of the anti-Hr type, produced by an Rh positive mother of an erythroblastotic infant. This agglutinin, they believe, may make possible in the laboratory, the differentiation of homozygous or heterozygous Rh factor bloods.

But on the other hand, the Hr factor can not be held responsible for all cases of isoimmunization in Rh positive mothers. Isoimmunization by the A and B factors must be considered, particularly if both parents are Rh positive or Rh negative, while the dominant A and/or B are present in the father's but not in the mother's blood.[18]

In any event, the demonstration of an agglutinin in the mother's serum which will agglutinate the cells of the father and of the affected infant, must not be overlooked in considering the cause of both early and late fetal deaths, as well as, the causative agent in definite cases of erythroblastosis, unexplainable by the Rh factor.

The Rh Factor in Transfusion Reactions

Isoimmunization denotes immunization within the same species; i. e., the individual immunized and the immunizing antigen must belong to the same species. It is reasonable, therefore, to assume that individuals receiving multiple blood transfusions may be subjects for isoimmunization. There must, however, be an antigenic difference in the blood of the donor and the recipient for this to occur. In blood which is otherwise compatible, this antigenic difference is found in the Rh factor. Just as the Rh factor has been proven to stimulate the production of the anti-Rh agglutinin in pregnancy, so also, during the course of multiple transfusions, the recipient will be stimulated to produce the anti-Rh agglutinins.

Earlier in this paper, several reports of hemolytic transfusion reactions due to the Rh factor were cited.

DeGowin has recently reported a series of 5,386 transfusions in which six reactions could be attributed to isoimmunity to the

Rh agglutinogen.[20] Of these, isoimmunity in four were attributable to multiple transfusions, two to pregnancies. In no case was isoimmunization established before the fourth transfusion. Of this group 399 received four or more transfusions. Approximately 15%, or 60, of these recipients should be Rh negative.

In a series of 2,528 transfusions in this institution, hemolytic reactions in two individuals were attributed to isoimmunization to the Rh antigen.

Case 1 (14950-O). A 39-year-old male, Blood Group O, was admitted to this hospital December 1, 1943, complaining of a "ruptured muscle in my leg." Taken to surgery, a tumor of the quadriceps femoris muscle was excised. The pathological diagnosis on section: rhabdomyosarcoma—malignancy Grade III. On December 15, the leg was amputated at the hip joint.

The patient received pooled plasma and blood as outlined below:

12-15-43—1000 cc. pooled plasma—no reaction.
12-15-43— 500 cc. Group O whole blood—Rh unknown—no reaction.
12-17-43— 500 cc. Group O whole blood—Rh unknown—no reaction.
5- 3-44— 500 cc. Group O whole blood—Rh unknown—no reaction.
5- 5-44— 500 cc. Group O whole blood—Rh unknown—no reaction.
5-10-44— 500 cc. Group O whole blood—Rh unknown—no reaction.
5-17-44— 500 cc. Group O whole blood—Rh unknown—no reaction.
6-12-44— 500 cc. Group O whole blood—Rh unknown—no reaction.
6-28-44— 500 cc. Group O whole blood—Rh positive, started. After 100 cc., patient had a severe chill, temperature elevated to 104°. Transfusion discontinued; no hemoglobinuria apparent. Patient's blood tested for Rh and found to be Rh negative. The anti-Rh could not be demonstrated.
7- 8-44— 250 cc. Group O Rh negative whole blood—no reaction.
7- 9-44— 250 cc. Group O Rh negative whole blood—no reaction.
7-16-44— 500 cc. Group O Rh negative whole blood—no reaction.
7-24-44— 250 cc. Group O Rh negative whole blood—no reaction.
7-25-44— 250 cc. Group O Rh negative whole blood—no reaction.
7-28-44— 500 cc. Group O Rh positive whole blood started; after 50 cc., patient began to chill; transfusion discontinued; temperature 101.4°.
8- 4-44— 500 cc. Group O Rh negative whole blood—no reaction.
8-11-44— 500 cc. Group O Rh negative whole blood—no reaction.
8-13-44—Patient expired. Death was due to the neoplasm.

This patient received eight transfusions of 500 cc. each, Group O whole blood before giving any evidence of immunization to the Rh factor. On 7-28-44, no Rh negative blood was available. Since the patient had no hemolytic symptoms after his first reaction, it was decided to try apparently compatible Group O, Rh positive blood. As given above, the patient reacted after 50 cc. of this blood.

Case 2 (9664N). On August 18, 1942, a female aged 52 was admitted to this hospital with a diagnosis of pernicious anemia. She complained of weakness, fatigue and tingling sensations in her extremities. Her blood count presented the following picture: Hb. 4.6 gms./100 cc., rbc 1.25, color index 1.33. She had had five pregnancies and had five living children, all of whom were normal at birth. She received the following transfusions:

8-19-42—500 cc. Group O Blood—Donor: patient's son; after absorbing the 500 cc. the patient developed a chill; temperature 103.4°.

8-20-42—500 cc. Group O Blood—Donor: patient's husband; no chill reported but patient displayed profuse diaphoresis.

8-24-42—500 cc. Group O Blood—Donor: patient's sister-in-law; all blood absorbed; one hour later patient had a chill; temperature 100.6°.

8-25-42—500 cc. Group O Blood—Donor: patient's daughter; patient had a chill; temperature 101.6°.

8-26-42—Count taken: Hb. 9 gms./100 cc., rbc 2.49.

8-27-42—Patient discharged from hospital by ambulance. Two specimens of urine received for analysis on 8-19 and 8-24, demonstrated no hemoglobinuria.

1-24-45—Patient re-admitted.

1-25-45—Count taken: Hb. 5.5 gms./100 cc., rbc 1.37.

1-25-45—500 cc. Group O, Rh positive blood started. After 200 cc. were absorbed, patient developed a severe chill lasting fifty minutes, accompanied by emesis of undigested food. Temperature, 104.6°. Blood discontinued at beginning of reaction. First specimen of urine obtained after reaction had a "smoky appearance" and gave a positive benzidine test though no rbc were seen microscopically. An Rh determination proved the patient to be Rh negative.

1-27-45—500 cc. Group O Rh negative blood given; no reaction.
1-30-45—Count taken: Hb. 8.5 gms./100 cc., rbc 2.06.
1-31-45—500 cc. Group O—Rh negative blood absorbed; no reaction.
2- 6-45—Count taken: Hb. 9.0 gms./100 cc., rbc 2.13.
2- 7-45—500 cc. Group O Rh negative blood absorbed; no reaction.
2-10-45—Count taken: Hb. 9.5 gms./100 cc., rbc 2.8. 500 cc. Group O Rh negative blood absorbed; no reaction. Patient discharged from hospital by ambulance.

Patient's husband:	Group O—Rh positive
♀ child	Group O—Rh positive
♀ child	Group O—Rh positive
♀ child	Group O—Rh negative
♂ child	Group O—Rh ? —recently killed in foreign service.

This patient, no doubt, was slightly immunized to the Rh factor, through her pregnancies. Her first transfusion twenty years after her last pregnancy, produced a mild reaction. Four transfusions of Rh positive blood were given before the patient had a truly hemolytic reaction during her fifth transfusion. All subsequent transfusions with Rh negative blood were without reaction.

Summary and Conclusions

1. The history, present knowledge, and nomenclature of the Rh factor in blood are briefly summarized.
2. The Rh factor in its relation to erythroblastosis fetalis and to multiple transfusions is discussed; case histories are presented.
3. It is demonstrated that a mother negative and a father positive to the Rh factor, do not necessarily mean absolute incompatibility; that normal offspring may derive from such a marriage.

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A REVIEW OF METHODS FOR DETERMINING THE CONCENTRATION OF PENICILLIN IN BODY FLUIDS*

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Recent widespread use of penicillin as a chemotherapeutic agent in treating infectious diseases has necessitated the development of assay methods for this antibacterial agent. The chemical formula for this antibiotic, if known, has not yet been released; and there is no accurate chemical test in which the determination of the level in body fluids is as simple and usable as our present methods for determining sulfonamide levels. Until such a test is available, the only methods used will be biological assays. It is obvious that, since the substance in question is antibacterial, the simplest test will be one in which the inhibitory or lethal effect of the unknown will be tested against strains of bacteria.

It has been suggested that certain bacteriological tests should be run in connection with penicillin treatment.[8] The first of these (to be determined before treatment is begun if possible) should be a species diagnosis of the organism causing the infection; and it would be desirable to determine the sensitivity of this particular strain to penicillin so that adequate levels of penicillin could be maintained in the body fluids. Following administration of pencillin, determinations of the level in body fluids should control the amount given. At present, most of this work cannot be done in a busy clinical laboratory; but from recent publications, it seems likely that a routine and simple method may be established.

Methods available at present for penicillin assay have been developed specifically for research and industrial laboratories where the culture fluid, extract, etc., are tested for potency. In almost

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every case the actual amount of penicillin per ml. is of the order of thousands of Oxford units. The most common methods are: (1) serial dilution method in liquid or solid medium using *Staphylococcus aureus* H (Fleming's original strain); (2) turbidimetric method, in which a standard curve is constructed from turbidimetric measurements of the bacterial growth in different concentrations of the unknown solution, and (3) the most commonly accepted method of cup-plate assay in which the diameter of the zone of inhibition around a cylinder (cup) of penicillin is used to determine the potency.[6, 7, 12] From the cup-assay method, the Oxford (or Florey) unit is defined as that amount of penicillin which will give a zone of inhibition against *Staphylococcus aureus* H equal to that of the original Oxford standard (24 mm. in diameter).[1]

Various modifications of the above methods for determination of the level in body fluids have been developed. Since the concentration in most body fluids (with the possible exception of urine) seldom exceeds 1 unit/ml. unless the test is done immediately after injection; a more critical method must be used to detect such small concentrations. Standard solutions of penicillin must be available in any assay procedure. These can be obtained from the United States Department of Agriculture, Northern Regional Research Laboratory, Peoria, Illinois, where the work is directed by Dr. R. O. Coghill. A National Standard, consisting of 3 gm. of pure crystalline penicillin has been set up by the Food and Drug Administration and the value established at 1,650 Oxford units. It seems likely that standardization will soon be based upon this standard.[2]

One of the first methods developed was the serial dilution method of Rammelkamp.[10] Dilutions of serum and dilutions of a standard solution of penicillin are set up with the usual precautions to maintain sterility. Red blood cells and an especially sensitive strain of hemolytic streptococcus are added, the entire set-up incubated and the end-point read as the tube containing the smallest amount of serum and penicillin which inhibited hemolysis of the red cells. By comparison with the standard solution of penicillin the concentration in 1 ml. of serum could therefore be computed. Values of hundredths of units can be determined. Recently this method has been modified by Stanford University workers[9] and compared with the cup-assay and turbidimetric method. Instead of using serial dilutions, simple tube dilutions (0.5 ml., 0.4 ml., etc.)

of the serum were used, and this method was found to be more suitable for routine clinical use. The standard solution contained 0.1 unit per ml. and the calculations are based on units per ml. rather than on the actual number of units in each tube as in the previous dilution methods.

Fleming published a method using the same system as an indicator, i. e. hemolysis of red cells by hemolytic streptococci.[4] The test was set up by the slide cell technique and by capillary tubes which were incubated over night. The test as described is sensitive to a 1/16 dilution of blood containing 0.5 units/ml.; i. e. to about 0.03 unit/ml. It is stated that this test is also accurate in the presence of other organisms (which is important in the presence of septicemia).

Another simple method which is essentially a modification of Rammelkamp's original dilution method has been developed by workers at the Jewish Hospital, Brooklyn.[11] A standard solution of penicillin, a standard organism (*Streptococcus pyogenes* C203) and the patient's serum are required. All tests are performed by adding standard and serum in proper dilutions to brain heart infusion broth (Difco) which are inoculated with the streptococcus culture. After 18 hours' incubation, the end point is read as that amount of penicillin and serum which will inhibit the growth of the streptococcus. The streptococcus strain used showed remarkable constancy in sensitivity to 0.01 unit of penicillin/ml. Calculations are as follows:

> Penicillin (in units) inhibiting test organism divided by amount of serum inhibiting test organism equals units per ml. serum.

Fleming's method has been recently modified so that the test is set up in test tubes and transferred to capillary tubes for incubation. Serial dilutions of standard penicillin and whole blood are set up in the usual manner, and these are inoculated with equal amounts of a culture of hemolytic streptococci. The end point is the most dilute control and unknown tube without hemolysis and bacterial colonies. This tube contains 1 unit of penicillin and calculations can be made for the amount present in 1 ml. of whole blood.[14]

It is interesting to note just exactly what concentration of penicillin might be expected in the blood of patients under treat-

ment. Fleming[5] found that on intravenous injection of 15,000 units: 4 units/ml. whole blood were present immediately; which in 10 min. fell to 0.5 units; in 1 hr. to 0.03 units and on a 3-hour test no penicillin could be detected. At the same time intervals after 15,000 units by intramuscular injection, 0.0, 0.12, 0.5 and 0.0 units were present. Subcutaneous injections gave similar results to the latter. Intramuscular injections of 100,000 units resulted in the maximum blood level of 2-3 units/ml. after 15 minutes with some still present after 5-6 hours. The intravenous drip method maintains a level dependent upon the rate of infusion. By dripping 240,000 units/24 hours intramuscularly for three days, a level of 0.5 unit/ml. was maintained. Workers have reported that most streptococci are sensitive to less than 0.1 unit/ml.[13] The size of the inoculum, different strains, and different serological groups of Lancefield do not appreciable affect this sensitivity.

Penicillin is inactivated by contact with human serum or blood;[3] and this inactivation is much greater at 37° C. than at lower temperatures. This inactivation may lead to underestimation of the amount of penicillin in a patient's serum and necessitates that the blood be tested as soon as it is taken. The inactivation at 37° C. is of the order of 5% per hour for an initial concentration of 2 units per ml.

Summary

The most common method of assaying penicillin for research and industrial work, the Oxford cup method is unsatisfactory for clinical determinations if the body fluid contains less than 1 unit/ml. The dilution method as introduced by Rammelkamp is accurate; but, because of the excessive pipetting, is laborious. Other more recent methods in which the dilutions are not serial, but are in decreasing amounts, simplify the determination in body fluids. The addition of red blood cells and the consequent use of hemolysis as an end point has been a valuable aid in reading these tests. The most effective use of penicillin in treatment necessarily involves the evaluation of dosage schedules. Dosage schedules at the present can only be determined if the susceptibility of the infective organism is known and if the body fluids levels can be determined. It is to be hoped that some simple chemical test will replace the variable and inaccurate bioassays now available.

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LABORATORY ASPECT OF THE SULFONAMIDES

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With the advent of the sulfonamide drugs as chemotherapeutic agents, an entirely new field of laboratory procedures was unfolded —new crystals found in microscopic urinalysis, sulfa determinations of blood and urine, consisting of both qualitative and quantitative tests. Then, too, because of their toxity, close check has to be kept of the blood picture and urine of these patients receiving the drugs to detect any evidence of anemia, leukopenia or kidney damage at the earliest possible moment.

These sulfonamides, which perform wonders in certain bacterial infections, are chemical compounds, drugs, which are effective not because of bactericidal powers but rather of bacteriostatic. They act by hindering multiplication of invading bacteria, thus holding them in check until the body's defenses have time to form an adequate defense against the invaders. It is well also to keep in mind that the patient gets well because his own defenses are able to rise to the occasion and develop a sufficient immunity to overcome the invading infection. The sulfonamides are not in themselves able to eradicate bacterial infection nor even to hold certain infections in check indefinitely. Supportive measures whose purpose is to keep the patient's general condition as good as possible and to assist in production of immunity are indicated.

An important chemical reaction which affects the therapeutic usefulness of the sulfonamides, occurs within the body, principally in the liver, and is known as acetylation. When conjugated with the acetyl radical, the sulfonamide generally appears therapeutically inactive in the body, and this acetylated form has a close relationship to the occurrence of toxic symptoms. The rate of acetylation seems to vary in the different individuals.

After administration, most of the drugs are absorbed rapidly

and appear in the blood, urine and body fluids in both forms, the free sulfonamide and the conjugated form. They are excreted almost entirely by the kidneys, essentially all of the free form being excreted in 24 hours, the acetylated in 48 hours. As a general rule the acetyl form is more insoluble; therefore, there is a tendency for it, more so than the free form, to precipitate out in the urine forming crystals. However, crystals of the free form are found also. Both types of crystal may appear in either acid or alkaline urine, but they seem to be more soluble in alkaline urine. They are "impure" crystals, their form depending largely upon the impurities present in urine and differing from those of the chemically pure compounds. Their presence may indicate only that the drug is being excreted. In any case, particularly when they show up in unusually large numbers, pathological changes in the urinary tract may be foreshadowed. These crystals are retained by the kidney in renal impairment, and they may attain sufficient size to cause an actual occlusion of the kidney, pelvis or ureter. It is, therefore, important that the crystals be recognized for what they are and not confused with other structures.

The crystals of each drug are characteristic of the respective sulfonamide.

Being the first to attain wide use in this country, sulfanilamide perhaps has been the subject of more investigation than any other member of this group. The crystals are not common except when the blood concentration of the drug is very high. Free sulfanilamide forms long, transparent and colorless rhombic crystals with a tendency to aggregate. Acetylsulfanilamide forms sheaves and radiating bundles of prismatic crystals.

Sulfapyridine crystals are stubby prisms, whereas, the more frequently occurring acetylsulfapyridine crystals are colorless transparent plates, modified to resemble flint arrowheads, small fish, bow ties, flower petals, or playing card diamonds. Occasionally, large, somewhat opaque, centrally bound sheaves, or rosettes of coarse needles are found.

With sulfathiazole, it is noted that the crystals of the free form appear as transparent and colorless hexagonal plates which may be indented at ends and show an envelope-like marking. The crystals of acetylsulfathiazole form sheaves of needles with central

binding, much like shocks of wheat. These may present all degrees of fullness up to a stage where they form two half-circles fused at the center.

In the case of sulfadiazine, it is found that the crystals of the free form are the more abundant. They form dark, dense, greenish globules with either fuzzy or clear edges. They resemble chestnuts or chinquapin burrs, while acetylsulfadiazine forms "shocks of wheat" with eccentric binding or asymmetric rosettes resembling "shells." This eccentric binding differentiates them from the central binding of the acetylsulfathiazole crystals.

Crystals of the free form of sulfaguanidine rarely appear, but acetylsulfaguanidine forms large, clear and colorless rectangular plates, often with bulging sides, or sheaves of fine needles somewhat resembling tyrosine crystals.

Sulfasuxidine crystals do not appear in the urine because of the slight absorption of the drug from the gastrointestinal tract and its great solubility in urine.

Since the sulfonamide crystals may suggest crystals of dicalcium phosphate, uric acid or tyrosine, a chemical test may be used for identification.

It has been observed that with Erlich's paradimethylaminobenzaldehyde reagent, solutions of sulfonamides give a greenish yellow color. However, this color will be given also by other substances, as urea and novocaine. To ascertain that the color is due to the sulfonamide and not an interfering substance, chloroform is added and if the greenish yellow color is absorbed by the chloroform, the test is a positive one for the sulfonamides. If not, then the color was given by the interfering substance.

Now when a person is taking a sulfonamide, it is necessary, in order for the drug to be effective, that an optimum blood concentration of the free (active) form be maintained. Too, it is important for the well being of the patient that an excess be not given. To keep a check of these two important factors, determinations of the concentration of the free and conjugated drug in the blood should be made. There is a simple technique for determining the free drug. It consists in diazotizing the free sulfonamide drug

in the laked protein free blood filtrate with nitrous acid and then coupling this diazotized product with N-(1-naphthyl) ethylenediamine dihydrochloride to produce a purple-red azo dye. The amount of this dye is directly proportional to the concentration of the sulfonamide in the blood. This dye is compared colorimetrically with a similarly treated standard. It is not necessary that the standard be prepared from the respective drug. However, if a different drug is used in preparing the standard, this must be taken into consideration and a conversion factor has to be used in calculating the results. In this way the concentration, the mgs of sulfonamide, in the blood is determined. The optimum blood concentration of the free form of sulfanilamide is 7-10 mgs./100 cc. of blood, sulfapyridine or sulfathiazole—4-6 mgs./100 cc. sulfadiazine—8-12 mgs./100 cc. sulfaguanidine—4-5 mgs./100 cc., and sulfasuxidine —1-2 mgs./100 cc.

In order to find out the amount of conjugated sulfonamide that is present in the blood, a determination of the free drug is made and a determination of the total amount of sulfonamide, and by subtraction of the free from the total the amount of conjugated is found. In determining the total amount, the same procedure just given is followed after the acetylated sulfonamide is converted to the free form. This is done by heating a portion of the protein free laked blood with hydrochloric acid.

If a quantitative determination of the sulfonamide in the urine is indicated, this may be done in the same way as above. However, before diazotizing it, the urine is diluted 1/50, 1/100, and 1/200 in an attempt to obtain a concentration between 0.5 and 2.0 mgs. of free sulfonamide/100 cc. of urine and it is then treated with hydrochloric acid.

In all of these ways it is possible to keep a complete laboratory picture of a patient's progress while being treated with the sulfonamides. And so we can see the part, and a very important one, that the laboratory plays in aiding the doctor in his treatment of patients with the sulfonamides.

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THE STUDY OF THE EOSINOPHILE IN ALLERGIC STATES*

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The study of the eosinophilic response is so very little used; and yet is packed full of information. It is hoped by this paper that you are encouraged in recommending its use to the physicians you contact.

If I had to list the studies used in allergic states in the order of their importance—they would be placed as follows:

1. Allergic history;
2. The search for eosinophiles in the nasal smear or blood;
3. Skin tests, etc. One should notice that skin testing is in third place.

Every patient with a nasal secretion, whether acute or chronic, seen by a physician should have a nasal smear, especially if long continued local or general therapy is suggested, or any operative procedure of the nose or throat is completed.

I should like to state three clinical cases which emphasize the last statements.

(1) D. M., age 11, presented herself in September, 1940, at our nose and throat clinic with enlarged tonsils for tonsillectomy. The allergic history was recognized and an allergic survey and control were instituted. Her nasal smear showed many eosinophiles and the blood eosinophilia was increased. Today she is much improved but still has her tonsils. They may be taken out as an elective operation.

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(2) W. H., age 4, was brought to the clinic with a history of "always being" in the hospital due to "severe colds" and convulsions. After an allergic survey including a nasal smear which revealed no eosinophiles and organisms four plus; bacterial vaccine was given with excellent results. We feel no respiratory vaccine should be administered before the allergic state is known and the presence of many organisms are demonstrated in the nasal smear.

(3) W. S., age 11, came to the office with a history of "severe sinus trouble." Several nose and throat operations did no good and may have made him worse. An allergic survey revealed a highly allergic individual. Eosinophiles in the nasal secretion and in the blood were positive. Allergic control has helped him a great deal and if it had been done earlier many operations may have been avoided.

Children were used in these examples to show that allergic manifestations usually start early in life and to emphasize that these patients should be treated immediately to contradict the time-worn statement that these children will "grow out" of their allergic symptoms.

A few statistics were gathered from our clinic cases at St. Francis Hospital to give you some idea how often these procedures are used.

Number of cases reviewed	90
Number on which nasal slides were taken	62

RESULTS

Eosinophiles only	6
Eosinophiles and Neutrophiles	10
Eosinophiles and Organisms	16
Eosinophiles, Neutrophiles and Organisms	12
Total with Eosinophiles present	44
Neutrophiles and Organisms	7
Organisms only	6
Neutrophiles only	3
Negative	2
	18
Number of differential blood counts	31
Eosinophilia (5% or over)	13

Is the Eosinophile an immunologic response? For many years

physicians have recognized that it will fluctuate with immunologic reactions. Writers have recognized three primary sources where the increase of eosinophiles might take place:

(1) The local shock tissue;

(2) The blood;

(3) In the bone marrow.

The best authorities at the present time believe that this increase takes place in the local tissue. In 1939 Salaris and Guarnari reported an ingenious experiment to prove this point.

Fifteen patients suffering from bronchial asthma or allergic rhinitis were tested intracutaneously with specific allergens. Blood was drawn from the finger-tip at five, twenty, forty and sixty minutes respectively after the onset of the positive cutaneous reaction and the percentage of eosinophiles was determined. An increase in eosinophiles was noted within five to twenty minutes. This increase was more pronounced in the blood taken from the arm on which the cutaneous testing was performed. This finding, according to the authors, is suggestive of the local origin of eosinophiles.

We usually think of studying smears from nasal or bronchial secretion or in the blood. In our hands the nasal smear is much more reliable than the sputum. Often in bronchial secretion an increase of eosinophiles will only denote sensitization to organisms present, and additional allergic study will be negative. Occasionally any tissue or secretion may be subjected to this study; as section of nasal polyps, mucus membranes, veriform appendix and other pathological tissues, sinus drainage fluids, stools or urine. One should especially look for eosinophiles in obscure symptoms where no organic etiology is demonstrated such as in idiopathic colites, etc.

An increased blood eosinophilia may happen in many disease processes as parasitic infestations, etc. One should never make the mistake of attaching any allergic significance to increased eosinophilia in the presence of any other disease associated with this phenomenon. Recently Atmar in the Journal of the American Medical Association reported a familial eosinophilia. Abnormal counts being recorded by the patient's two brothers, two sisters and two children. An allergic patient may show a normal picture,

slight or marked eosinophilia. We consider 5% or above as an increase. In our patients with increased eosinophilia the counts ranged from 5% to 20% and rarely up to 30%. In a differential smear for eosinophiles one should count at least one hundred cells and, better still, fifty cells in each corner of the smear counting in zig-zag fashion. As many allergic individuals, without infection, show a rather low total white count, an edge along the smear helps speed up the counts.

Methods of Obtaining Nasal Smears

1. One might have patient blow nose into wax paper or cleansing tissue.

2. Swab-stimulation, if the nasal mucosa is fairly dry. This method is used by many but is not too successful.

3. From material taken from the pharynx in cases of post-nasal drip.

4. From sputum where bronchial secretion is present.

The first method is preferred as the patient often has a large amount of secretion, but not just when one looks into the nose. The patient is given a packet of two clean slides, toothpicks and cleansing tissue with instructions to make two slides at different times. The slides are brought to the clinic at the next visit. If the patients are well insructed this method is highly successful.

Remember one slide is a study at one particular time and as conditions change repeat slides are essential. Hansel recommends following changing conditions by repeated examinations. Both sides of nose might even be different, etc.

Preparation of Slide and Staining

Giemsa, Hansel's or Wright's stain may be used. The Giemsa and Hansel's give slight superior staining but after handling about three thousand slides, experimenting with many technics and using many stains, it has been decided for the sake of simplicity and time saving to stick to Wright's. It is hoped that in the near future a simple stain may be developed which is a combination of both

Wright's stain and Gram's. This would give differential staining of bacteria and the cells at one operation.

The nasal smear should be prepared and stained in the following manner. The secretion is spread thin on the slide and allowed to dry in clean air. After completely dried, the Wright's stain is applied and then distilled water. Then wash with distilled water. As a general rule the staining times are one-half the times as used for blood smears. Stand on end or dry over a small electric bulb. Do not blot.

Examine under the oil-emersion lens.

At a risk of making my remarks somewhat boring it is necessary to emphasize some of the characteristics of the eosinophile.

1. It is larger than the neutrophile.

2. The membrane is fragile so many broken cells are seen and also scattered loose granules. These granules are orange-red while organisms are purple.

3. There are large acidophilic granules staining brilliant orange-red with Wright's.

4. The nuclei usually are two in number and stain blue.

5. The cytoplasm is soaked with granules.

Do not call cells eosinophiles unless they meet these criteria. Sometimes one sees many cells with small granules stained a brilliant orange-red. These cannot be called eosinophiles or neutrophiles. They are listed as transitional forms.

Interpretation

The eosinophiles may be very unevenly spread, all clumped in a mass of mucus. This mucus often interferes with good staining. Long study may be essential. Often there may be one clump in the entire slide which shows hundreds of eosinophiles.

When studying, all the elements in the smear should be observed and recorded. This goes further than Hansel but has definite advantages. He reports only eosinophiles and neutrophiles.

With some experience organisms usually can be identified; they stain blue or purple with Wright's stain. The type can often be

guessed successfully. When the slide is loaded with organisms a Gram's stain is done and a bacteriologic study of the secretion is obtained.

Epithelial cells do not usually signify anything pathological but a sarcoma of the Maxillary Antrum was once encountered, which showed many sarcomatous cells on Wright's stain of the nasal secretion.

MAJOR TYPES OF RESPONSES

1. *Allergic*

E++++	N 0	Or 0	Ep+

2. *Infectious*

E 0	N+++	Or+++ (Predominating) (? diplococci)	Ep+

3. *Bacterial allergy or a secondary infection on top of an allergic response.*

E++	N+++	Or+++	Ep++

E—Eosinophiles
N—Neutrophiles
Or—Organisms
Ep—Epithelial cells

A rare eosinophile, especially in children, can be regarded as normal. An occasional neutrophile or lymphocyte is normal. Hansel states, "In evaluating the number of neutrophiles in a secretion one must take into consideration that the neutrophilic response is always greater than the eosinophilic response and that the number of neutrophiles usually outnumber the eosinophiles about ten to one, therefore, a plus-minus or a plus-one of neutrophiles represents about ten times as many eosinophiles. In a smear with four-plus neutrophiles the field is completely covered with them."

In conclusion, I have attempted to point out the diagnostic importance of the eosinophile in allergic states and have given aids in the preparation and interpretation of smears taken.

A REPORT ON THE STATUS OF THE BILL FOR COMMISSIONING MEDICAL TECHNOLOGISTS IN THE ARMY OF THE UNITED STATES AND IN THE NAVAL RESERVE

The status of the Bill H.R. 3147 is, I am sure, a question of great interest to each of us. First, may we pause to thank Mrs. Evelyn Jardine for her most untiring efforts in behalf of the bill. None of us, I am sure, can quite realize the many hours she has spent in working out the numerous details necessary to bring it to the point it reached. And to each of the groups and individuals who did their part in calling it to the attention of those who would have the power to vote upon it, go our appreciation and thanks.

Below is a paragraph from the letter to Mrs. Jardine from the Honorable Sherman Adams, M.C., who introduced the bill in the House of Representatives: "In the face of the opposition of both Departments (War and Navy) to this legislation, it will take great pressure from members of Congress in general to progress this bill. At the moment I do not know from where the pressure will be forthcoming."

From the Secretary of War to the Hon. Andrew J. May, Chairman, Committee on Military Affairs, House of Representatives, the following is quoted: "The War Department is opposed to the enactment of H.R. 3147, 79th Congress, a bill to authorize the appointment of qualified medical technologists as commissioned officers in the Army of the United States and in the Naval Reserve." Here follows the body of the bill, including a quotation of the proposed requirements for the commission:

"Medical technologists include laboratory workers who perform various types of laboratory tests, such as blood counts, urinalyses, etc. Much of this work is of a purely routine nature and those performing it do not necessarily possess the background or experience which would qualify them for commissions. Where duties

involve supervision or the exercise of greater responsibilities, existing law and regulations authorize their commissioning in the Sanitary Corps which was organized in the Medical Department to provide highly trained specialists in the fields of parasitology, bacteriology, biochemistry, serology, and certain other professional specialties. Approximately twenty-seven hundred officers have been commissioned in the Sanitary Corps and the most recent procurement objective for specialists in the laboratory field was completed in February, 1945. At the present time, the need for such officers has been amply filled. In fact, with redeployment and readjustment of the Army's strength, it is probable that some of these officers will be permitted to return to civilian life.

"The proposed standards of education and experience for commissioning medical technologists are much lower than those now required for appointment in the Sanitary Corps. If lower standards are established by law for the appointment of medical technologists as commissioned officers, it will tend to undermine the high standards which now exist for appointment in the Sanitary Corps generally.

"The War Department is unable to estimate the cost of the proposed legislation.

"For the foregoing reasons, the War Department recommends that H.R. 3147 be not favorably considered.

"The Bureau of the Budget advises that there is no objection to the submission of this report."

Signed Henry L. Stimson, Secretary of War
August 3, 1945

From the Secretary of the Navy, we have the following report:

The substance of the bill is quoted and then: "It will be thus noted that the requirements for registration do not exceed two years of formal education in a recognized college or university plus one year in an approved training school for medical technologists, or the equivalent period as an apprentice. Registration is possible in some cases with no, or but one year of, formal education plus the necessary professional experience.

"Appointment to officer status in the Medical Department of the Naval Reserve, under existing regulations, are limited to pro-

fessionally qualified individuals with at least four years of collegiate training or who have a degree from an accredited institution of higher education.

"By the enlistment of medical technicians from civilian life and by the continued use of present naval facilities for training hospital corpsmen, it is anticipated that it will be possible to supply a sufficient number of qualified personnel to fulfill the needs of the Hospital Corps of the Navy for technicians.

"At the present time, medical technologists possessing the educational and professional qualifications required by current regulations are eligible for consideration for appointment in officer status. Women so qualified have been appointed in Class W-V(S) and are now on duty as medical technicians, pathologists, bacteriologists, parasitologists, etc. Also the Hospital Corps at present includes officers who have been certified by the Registry of Medical Technologists and others, who, no doubt, possess the qualifications herein stated. Such officers are assigned to billets as laboratory technicians as required.

"It should be noted that the proposed bill makes any person who has had two years of collegiate education, which includes the required courses outlined, plus one year of apprenticeship, eligible for appointment in officer status. Certification by the Registry of Medical Technologists would not be required under the provisions of the bill.

"It is the opinion of the Navy Department that the enactment of subject bill is not required for any Naval purpose and the Navy Department recommends against the bill for the following reasons:

(a) The requirements for recognition as a medical technician are considerably below those now in force for appointment to officer status in the Naval Reserve.

(b) The needs of the Medical Department of the Navy for medical technicians are amply supplied by personnel in an enlisted status through the present methods of recruiting and training.

(c) Sufficient numbers of officers qualified in the various branches of medical technology are presently on duty or are available to fill all billets requiring such personnel.

"The Navy Department has been advised by the Bureau of the Budget that there would be no objection to the submission of this report."

Signed John L. Sullivan, Acting
August 7, 1945

So, from the above three letters, we may sum up that although we made the effort, it was not enough. However, we can derive the satisfaction that there are a few more persons who have heard of the profession of Medical Technology. We are shown, too, that we must make still greater efforts, individually, and through our local, state, and national organizations, so to encourage higher educational standards, that, in the future, there will be no question of the qualifications of medical technologists being sufficient to give him as high a rating as persons in kindred professions.

Rose Matthaei, President.
ASMT

ABSTRACTS

TRIPLE-SUGAR IRON AGAR MEDIUN FOR THE IDENTIFICATION OF THE INTESTINAL GROUP OF BACTERIA: A. A. Hajna, Jr. Bact., Vol. 49, No. 5, May, '45, p. 516.

The proposed medium is said to give more clear-cut reactions for acid and gas and show greater sensitivity for H_2S than those in common use. The formula is as follows:

Distilled water	1000 ml.
Agar (dry)	13 g.
B.B.L. nutripeptone	20 g.
(Bacto-peptone 15 g. plus bacto-proteose peptone 5 g. may be used)	
Sodium chloride	5 g.

Dissolve in the Arnold sterilizer or in flowing steam in the autoclave. When melted, adjust to pH 7.5.

Admix:

Lactose	10 g.
Sucrose	10 g.
Glucose	1 g.
Sodium thiosulfate	0.2 g.
Ferrous ammonium sulfate	0.2 g.

The pH now should be 7.4.

Lastly add:

Phenol red (1% aqueous)	2.5 ml.

Dispense in 5 ml. portions. Autoclave at 12 lbs. for 15-17 minutes.

STORAGE OF INFLUENZA VIRUS FOR USE IN TYPING CLINICAL CASES: A. L. Florman and A. B. Weiss, Jr. Bact., Vol. 49, No. 5, May, '45, p. 507.

This study was undertaken to determine the most satisfactory manner in which influenza virus might be supplied to hospitals in event of outbreaks. Test material consisted of allantoic fluids con-

taining influenza A(PR-8) and B(Lee) viruses. Preservatives tested were 0.2% formaldehyde, 0.2% sulfanilamide and 33% buffered glycerol. Glycerin and sulfanilamide were satisfactory while formaldehyde, in the concentration used, destroyed the virus in a short time. Development of turbidity and presence of contamination did not interfere with the hemagglutinating reaction. However, E. coli and some other bacteria are also capable of agglutinating RBC and thus contamination with these might interfere. Use of 0.2% sulfanilamide and icebox storage are recommended.

A MEDIUM CONTAINING AN ACID CASEIN HYDROLYZATE FOR USE IN TESTING DISINFECTANTS: P. A. Wolf, Jr. Bact., Vol. 49, No. 5, May, '45, p. 463.

A liquid medium is described in which an acid-hydrolyzed casein and uracil constitute the main sources of nitrogen. Thiamine hydrochloride and niacinamide are added to stimulate growth.

It was found that cultures grown in this medium had resistance equal to or greater than that of cultures grown in Food and Drug Administration broth. This resistance could be maintained with daily transfers in this broth much longer than in the FDA broth. Resistance to phenol remained more constant and was apparently uninfluenced by different lots of casein hydrolysate.

BACTERIOLOGICAL STUDIES IN ENDOCARDITIS: W. J. MacNeal and A. Blevins, Jr. Bact., Vol. 49, No. 6, June, '45, p. 603.

At least three blood samples on successive days and at different times of the day are suggested in endocarditis. If an antibacterial treatment has already been given the patient, an inactivating agent should be used in the cultures.

This report contains observations on 36 positive blood cultures. Of these 13 were Strep. salivarius, 7 were Strep. equinus, 6 were Strep. faecalis, 3 were Strep. bovis, 2 were in the viridans group unspecified, 2 were in the micrococcus group unspecified, 1 was S. aureus, 1 was an actinomyces and 1 a Gram negative rod.

The organisms in the salivarius and equinus groups were very susceptible to penicillin. The organisms in the faecalis group did not respond even to massive doses of penicillin but were very susceptible to lysis by bacteriophages. The organisms in the miscellaneous group varied in their behavior toward penicillin.

CONGENITAL THROMBOPENIA: A. E. Schefrin and A. Schechtman, Am. Jr. Dis. Children, Vol. 70, No. 1, July, '45, p. 29.

This is a description of a case of congenital thrombopenia. It is unusual in that the blood of the mother showed no tendency to purpura and there was no family history of it. The baby showed petechiae and a low platelet count.

A MICROCOCCUS PRODUCING A MULBERRY-COLORED PIGMENT: L. F. Holmes and M. E. Wilson, Jr. Bact., Vol. 49, No. 3, Mar., '45, p. 311.

Morphological and cultural characteristics of a micrococcus isolated from wounds are given. It is considered as a contaminant in the wounds. It is unusual in that it produces an opaque white growth on agar but a mulberry-colored growth on potato slants. It is hemolytic and coagulase-negative.

STREPTOMYCIN: ABSORPTION, DIFFUSION, EXCRETION and TOXICITY: D. H. Heilman, F. R. Heilman, H. C. Hinshaw, D. R. Nichols and W. E. Herrell, Am. Jr. Med. Sci., Vol. 210, No. 5, Nov., '45, p. 576.

Administration by continuous intravenous drip appears to be the best way to maintain constant blood concentrations. If given intermittently, intravenous administration appears no better than intramuscular or subcutaneous administration. Intrathecal administration as well as oral administration and administration by nebulization have been successful.

Diffusion into the cerebrospinal fluid was not found except in cases of meningitis when the concentration equalled approximately one-fifth that of the blood serum. Streptomycin was transmitted through the placenta. At times the concentration was as great in cord blood as in the maternal.

Evidence indicates that streptomycin is concentrated and excreted in the bile. About ½ to ¾ of the total amount administered is usually recovered from the urine in the first 24 hrs.

Mild local skin reactions, chills and fever, joint pain and nausea have been reported from this drug. These may be due to the impure form that is now available rather than to the drug itself. There is no conclusive evidence to indicate interference with renal function, liver function or the hemolytopoietic system.

A PARACOLONLIKE BACILLUS ISOLATED FROM COLITIS IN AN INFANT: P. R. Edward, Jr. Bact., Vol. 49, No. 5, May, '45, p. 513.

This organism was isolated from the stools of an 11-month old baby suffering from acute colitis. It is considered a member of the paracolon group even though this strain did not ferment lactose because its antigenic composition places it within this group. Reactions of this type make classification of the enteric organisms difficult.

THE REACTION OF THE RETICULO-ENDOTHELIAL SYSTEM IN EXPERIMENTAL AND NATURALLY ACQUIRED BRUCELLOSIS OF SWINE: I. W. Brown, W. D. Forbus and G. P. Kerby, Am. Jr. Path., Vol. 21, No. 2, Mar., '45, p. 205.

Brucella suis isolated from a case of Hodgkin's disease was shown to be pathogenic for hogs though its pathogenicity was less than that of a strain isolated from a naturally infected hog. The reticulo-endothelial system, especially the lymph nodes, seemed to be most affected. Lymph nodes have been found to harbor the organism long after inoculation. The reaction of the reticulo-endothelial system of the hog in brucellosis is very similar to that of man in Hodgkin's disease.

EOSINOPHILIC LUNG: P. J. Hodes and F. C. Wood, Am. Jr. Med. Sci., Vol. 210, No. 3, Sept., '45, p. 288.

This disease is a pseodotuberculosis which is found in India and Australia and, since it is known to develop in Europeans, may affect returning American troops. The onset is characterized by lassitude, loss of appetite and slight fever. Later on asthmatic type of cough appears. This tends to become chronic and may at times result in severe asthmatic attacks. These symptoms may also be entirely absent. The white blood count is usually above 20,000 and may go to 80,000. The elevation is due to eosinophilia which may be as great as 92%. Mild secondary anemia as well as a moderately accelerated sedimentation rate may accompany the disease. Treatment with neoarsphenamine has been very effective. Two case histories are given.

BOOK REVIEW

IN THE DOCTOR'S OFFICE—THE ART OF BEING A MEDICAL ASSISTANT: Esther Jane Parsons. J. B. Lippincott Co., publishers, Philadelphia, Pa. Oct., 1945. Pp. 295. Price $2.00.

"How can I best serve the doctor and his patients?" Receptionists, medical secretaries, nurses and laboratory technicians who have asked this question—*and doctors who would like the question answered for their own assistants*—will find the information they seek in this new and helpful book. It shows, in a lucid, interesting manner, the many ways in which medical assistants can increase their personal efficiency, and thus prove more valuable to their profession.

Miss Parsons, a former medical assistant and teacher of assistants, has drawn upon her wide and practical knowledge of this highly specialized field to present one of the most concise, yet complete guides ever offered on the subject. Much of the material in this useful book is based on a series of lectures given by the author to medical assistants in training. It contains not only the author's personal observations, based on experience, but practical advice and suggestions actually submitted by doctors.

NEWS AND ANNOUNCEMENTS

Annual Convention
A. S. M. T.
San Francisco, Calif.
July 1, 2, 3, 1946

ANNOUNCEMENT FROM THE 1946 PROGRAM COMMITTEE

Dear American Society Members:

Plans are under way for the 1946 American Society of Medical Technologists' Convention in June and it is earnestly hoped that there will be ample material submitted for the program.

During these "war years," no doubt many of you have done some outstanding work or discovered new techniques for old tests —June would be a good time to pass along this new knowledge and no better place than at the convention.

As a reminder, here are a couple of the important rules applying to the programs:

(a) Only members of the American Society of Medical Technologists are eligible for awards, but papers from non-members are welcome, too.

(b) Scientific papers should be limited to 20 minutes.

Maybe you have not an article to submit, but know of someone who has, but just needs a little coaxing. Won't you let us have the name and address and we will gladly do the coaxing.

So let's get started on those scientific papers—NOW!!

Watch the next issue for more announcements.

The Program Committee: Mrs. Kathleen Cornell Bradley, M.T. (ASCP), chairman, 1340 W. Huntington Park Ave., Philadelphia 40, Pa.; Miss Elizabeth O'Toole, M.T. (ASCP), 1345 Elati St., Denver, Colo.; Miss Elizabeth Pitsch, M.T. (ASCP), Sharp Bldg., Lincoln 8, Nebraska.

ANNOUNCEMENT FROM THE NOMINATING COMMITTEE

To the Members of the A.S.M.T.:

Again it is time to select a new slate of officers for the year 1946-37. The Nominating Committee is truly anxious to select capable officers representative of the entire membership.

In order to do so, we are asking the cooperation of the members to suggest to us names of persons whom they feel are qualified for office. Along with the name or names, please mention the qualifications of each person suggested; that is, the part he or she has played in his or her local, state or national organization.

Please mail all suggestions to one of our committee members no later than March 1, 1946.

We shall appreciate these suggestions and each shall be carefully considered. We hope that with your assistance, we may present a slate of officers at the June Convention that will be a credit to our American Society.

Sincerely yours,

Clara T. Kruse, M.T.
1433 Benton Street
Rockford, Illinois
Rachel Lehman, M.T.
Indiana University
Indianapolis, Indiana
Doris Bradway, M.T.
White & Johnson Rds.
Norristown R. D. No. 4, Pennsylvania

Nominating Committee, A.S.M.T.

PAPER WRITING CONTEST NOTICE

Two cash prizes of ten dollars and five dollars respectively for the best and second best papers are being offered by each state having a society for medical technologists, to active members in the

society for papers written on any phase of medical technology the author wishes to choose. The Committee on Awards will be three representative pathologists under the chairmanship of a medical technologist appointed for this purpose by the state. The two prize papers of each state are to be printed in the state journal and are to be forwarded to Mrs. Kathleen Cornell Bradley, 1340 Huntington Park Avenue, Philadelphia 40, Pennsylvania, chairman of the committee of the 1946 annual convention of the American Society of Medical Technologists. Of these, the two best papers will then be subject to further awards by that committee and will appear in *The American Journal of Medical Technology*.

Other papers of merit will receive honorable mention in the *Journal*.

The contest for each participant begins with the reading of *this* notice and closes the third week in February, 1946.

On Writing Papers: Essentially the whole problem of writing papers resolves itself into the WHOM, WHY, HOW, and the RESULTS. A paper written with such simple factors as guides becomes a contribution to the chosen field—a human document which has gotten away from being a treatise on what others have accomplished; and has also gotten away from being a pretense to a research problem. A paper written under the above guiding principles does not exhaust the field—it makes an interesting contribution and stimulates both the writer and the reader. It differs from the true meaning of research as a lecture differs from a seminar. It is a lecture that makes a seminar possible. It is a paper like this that makes research possible.

Propose to yourself your theme and select the title. Read all that is printed in the field to get acquainted with what other people have had to say about your problem. Include in your reading unitive work that is particularly stimulating to you. Now begin writing and write as you would talk—at white heat. Set aside your paper and let it cool. Go over it for organization, looking to see that you actually said what you wanted to say, and in the most interesting manner possible. See that there is an introduction, possibly a brief review of the literature, a discussion in good sequence

of thought with illustrations and definitions of technical terms where necessary; a summary, and your conclusions. Keep your verbs in the same tense; be very careful about spelling, and whatever details of setting down your expressions you adopt be consistent throughout your paper. Wherever references to something read is made, due acknowledgment of the source must appear either as a footnote or at the close of the paper.

For helps in spelling hyphenated words, for capitalization, and writing references to sources, and for many other details the following book is very helpful: "A Manual of Style," by the University of Chicago Press, Chicago, Illinois. It may be gotten for $3.00 and when ordering specify the tenth edition.

BOARD OF REGISTRY AWARD

The Board of Registry is pleased to announce that prize of fifty dollars will be awarded to the registered Medical Technologist giving the best paper at the next annual meeting of the American Society of Medical Technologists. May 1 is the deadline for acceptance of papers. For full particulars write to: Miss Hermine Tate, Executive Secretary, American Society of Medical Technologists, Medical Arts Building, Lafayette, Louisiana.

NEWS FROM EXECUTIVE OFFICE

Due to the continuous change of address of many A.S.M.T. members, we feel that it is impractical to print a roster in this issue of the Journal.

However, we are happy to tell you that the membership has increased from 1,100 members in January, 1945, to 1,555 members as of December 15, 1945.

There are 223 applications approved, and pending payment of dues for completion of membership. There are new applications coming in daily.

At one time there were 85 A.S.M.T. members serving in the armed forces, although many of these have returned to civilian life now.

Distribution of the membership by states is as follows:

Alabama	12	New Jersey	32
Arizona	10	New Mexico	9
Arkansas	21	New York	81
California	96	North Carolina	24
Colorado	29	North Dakota	10
Connecticut	10	Ohio	52
Delaware	6	Oklahoma	52
Dist. of Columbia	17	Oregon	17
Florida	35	Pennsylvania	113
Georgia	10	Rhode Island	1
Idaho	9	South Carolina	9
Illinois	111	Nebraska	24
Indiana	52	Nevada	3
Iowa	24	New Hampshire	17
Kansas	25	South Dakota	10
Kentucky	42	Tennessee	29
Louisiana	26	Texas	123
Maine	6	Utah	7
Maryland	21	Vermont	3
Massachusetts	35	Virginia	28
Michigan	43	Washington	18
Minnesota	78	West Virginia	14
Mississippi	9	Wisconsin	55
Missouri	51	Wyoming	3
Montana	10	Foreign	33

Hermine Tate, M.T. (ASCP),
Executive Secretary.

Miss Marian Autch, M.T. (ASCP), 870 Lafayette Ave., Buffalo, N. Y., has been appointed Counsellor for the North Atlantic States, New York and New Jersey. Miss Esther Anderson, M.T. (ASCP), Riverside County Hospital, Arlington, California, is Counsellor for the Western States consisting of Utah, Nevada and California. Counsellors for other states were announced in the November Journal.

The Harris County (Texas) Society of Medical Technologists has just completed the mailing of approximately 10,000 booklets to each registered M.T. (ASCP).

STATE AND LOCAL SOCIETIES

The American Society of Medical Technologists now have twenty (20) affiliated societies.

MINNESOTA

Four regional meetings are planned for the medical technologists of the State of Minnesota. Two have already occurred and plans for the other two are pending.

The first of these regional meetings was held Dec. 7, 1945, in Hibbing, Minnesota, at the Hibbing General Hospital, under the local chairmanship of Miss Margaret Bateman, head technologist at that hospital. Medical technologists from Virginia, Eveleth, Buhl, Hibbing, Grand Rapids and neighboring towns attended that meeting.

The second occurred Dec. 8, 1945, at St. Cloud Hospital, St. Cloud, Minnesota, with Sr. M. Benora, OSB, as chairman of local arrangements committee. Medical technologists from Breckenridge, Fergus Falls, Clinton, Glenwood, Aitkin, Hugo, Little Falls, Willmar, Litchfield, Buffalo, Anoka, North Branch, Stillwater and other neighboring points attended this meeting.

A local society of medical technologists with the tentative title Central Minnesota Society of Medical Technologists was organized. The following officers were elected: President, Miss June Tyson, St. Joseph's Hospital, Brainerd; Vice President, Sr. M. Helen, OSF, St. Francis Hospital, Breckenridge; Secretary-Treasurer, Sr. M. Michael, OSB, St. Cloud Hospital, St. Cloud; President-elect, Mrs. Agatha Casey Caylor, Litchfield, Minn.; Board of Director Members: Miss Sylvia Finsand, Glenwood; Sr. M. Emerita, OSF, St. Gabriel's Hospital, Little Falls.

The Committee on Awards for Paper Writing Contest in Minnesota are the three pathologists: Dr. W. A. O'Brien, Minneapolis; Dr. Kano Ikeda, St. Paul, and Dr. A. H. Wells of Duluth with Sister M. Alcuin, OSB, College of St. Scholastica as the medical technologist representative and chairman.

ARKANSAS

Society Name	Chartered	Meetings	Secretary's Address
Arkansas Society of M.T.	National	Quarterly	Ann Snow, M.T. 216 East D. Ave., Park Hill, N. Little Rock, Ark.
Little Rock Society of M. T. Pres.—Lila Church, M. T.	State	Quarterly	Mrs. Chas. Wilkens, M. T. Baptist State Hospital N. Little Rock, Ark.

CALIFORNIA

Santa Barbara Chapt. of M. T. Pres.—M. Royal, M. T. (ASCP)	None	Monthly	Mary C. Ryan, M. T. Santa Barbara Gen. Hospital Santa Barbara, California

COLORADO

Colorado Society of M.T. Pres.—Mabel O. Stewart	National	Monthly	Annalee Breslford, M.T. 1560 High Street Denver, Colorado

DISTRICT OF COLUMBIA

District of Columbia Society of M.T. Pres.—Zanerian E. Funk	National	Monthly	Evelyn F. Ballou, M.T. 4105 Third Street N.W. Washington, D. C.

GEORGIA

Savannah Society of M.T. Pres.—Sadie Cartwright	National	Monthly	Jurelle S. Hooper, M.T. 20 East 56th Street Savannah, Georgia

ILLINOIS

Illinois Society of Clinical Laboratory Technicians Pres.—Fannie Warnock	National	Semi-annual	Edna H. Murmann, M.T. 3924 N. Monticello Ave. Chicago, Illinois
Chicago Society of M.T. Pres.—Helen E. Sunderland	National	Monthly	Joyce James, M.T. 939 North LaSalle Street Chicago 10, Illinois

INDIANA

Indiana State Society of M.T. Pres.—Virginia Sue Alley	National	Annual	Hazel Childs, M.T. Indianapolis City Hospital Indianapolis, Indiana
Indianapolis Society of M. T. Pres.—Mrs. Earl Lynn	National	Monthly	Miss Helen Kottolowski 1230 Villa Ave. Indianapolis, Indiana

KENTUCKY

Kentucky Society of M.T.	National	Monthly	Dorothea Distler, M.T. 860 Eastern Parkway Louisville, Kentucky

LOUISIANA

Louisiana State Society of M. T. Pres.—Eola Kendrick, M.T. (ASCP)	National	?	Elizabeth Norsworthy Touro Infirmary New Orleans, Louisiana
Caddo Parrish Society of M. T. Pres., Harriett Cypert	State	Monthly	Miss Alice Harkins

MASSACHUSETTS

Worcester District of M. T. Pres.—Marjorie Inman	National applied for	Monthly	Jane C. Zalesda, M. T. 16 Montrose St. Worcester, Mass.

MINNESOTA

Minnesota Society of M.T. Pres.—Esther Willbrecht	National	Annual	Margaret Strane, M.T. Miller Hospital St. Paul 2, Minnesota

NEBRASKA

Nebraska Society of M.T. Pres.—Romona Forbes	National	Annual	Marjorie Lundeen Lincoln General Hospital Lincoln, Nebraska
Omaha-Council Bluffs Society of M.T. Pres.—Rosemary McLennon	None	Bi-monthly	Elilie Blazek, M.T. (ASCP) 1469 So. Sixteenth St. Omaha, Nebraska

NEW HAMPSHIRE

New Hampshire Society of M.T. Pres.—Sister Marie-Rose (Larivee)	None	Annual	Marion P. MacMartin, M.T. Mary Hitchcock Mem. Hosp. Hanover, New Hampshire

NEW JERSEY

New Jersey Society of M.T. Pres.—Phyllis Stanley	None	Annual	Elsie Emmel, M.T. 304 Washington St. Glen Ridge, New Jersey

NEW YORK

Niagara Frontier Assn. of M.T. Pres.—Margaret Moore	National	Monthly	Alice Sprague, M.T. 110 Merrimac Buffalo 14, New York

OHIO

Ohio Society of M. T. District 1 Pres.—Lois Jane Scheffler	National	Annual	Mrs. Betty Milden City Hospital Akron, Ohio

OKLAHOMA

Society Name	Chartered	Meetings	Secretary's Address
Oklahoma State Society of M.T. Pres.—Oscar E. Stewart	National	Semi-annual	Hazel Clay, M.T. 514 North West 24th Street Oklahoma City 3, Okla.
Oklahoma City Society of M.T. Pres.—Vernal Johnson	State	Monthly	Dorothy Foreman, M.T. Universal Hospital Oklahoma City, Oklahoma
Tulsa Round Table of M.T. Pres.—H. L. Spencer	State	Bi-monthly	Mrs. Eliz. Goltry Johnson 401 Medical Arts Bldg. Oklahoma City 2, Okla.

PENNSYLVANIA

Pennsylvania Society of M.T. Pres.—Elsa Lynch	National	Monthly	Ann Caverly, M.T. 5000 Pulaski Ave. Philadelphia 44, Pennsylvania
Western Pennsylvania Chapter of P. S. M.T. Pres.—Helen Brumbough	State	Monthly	Dorothy Flohr, M.T. 308 Lavina Ave. Mt. Lebanon, Pittsburgh, Pa

TENNESSEE

Davidson County Society of M. T. Pres.—Thelma Wenzel	National	?	Mary Chunn Medical Arts Bldg. Nashville, Tenn.

TEXAS

Texas Society of M. T. Pres.—Margaret Davis	National	Annual	Miss Florence Petty Rusk, Texas

WASHINGTON

Inland Empire Society of M.T. Pres.—Frances Premo	None	Annual	Lenore De Vor East 547 Gordon Spokane, Washington

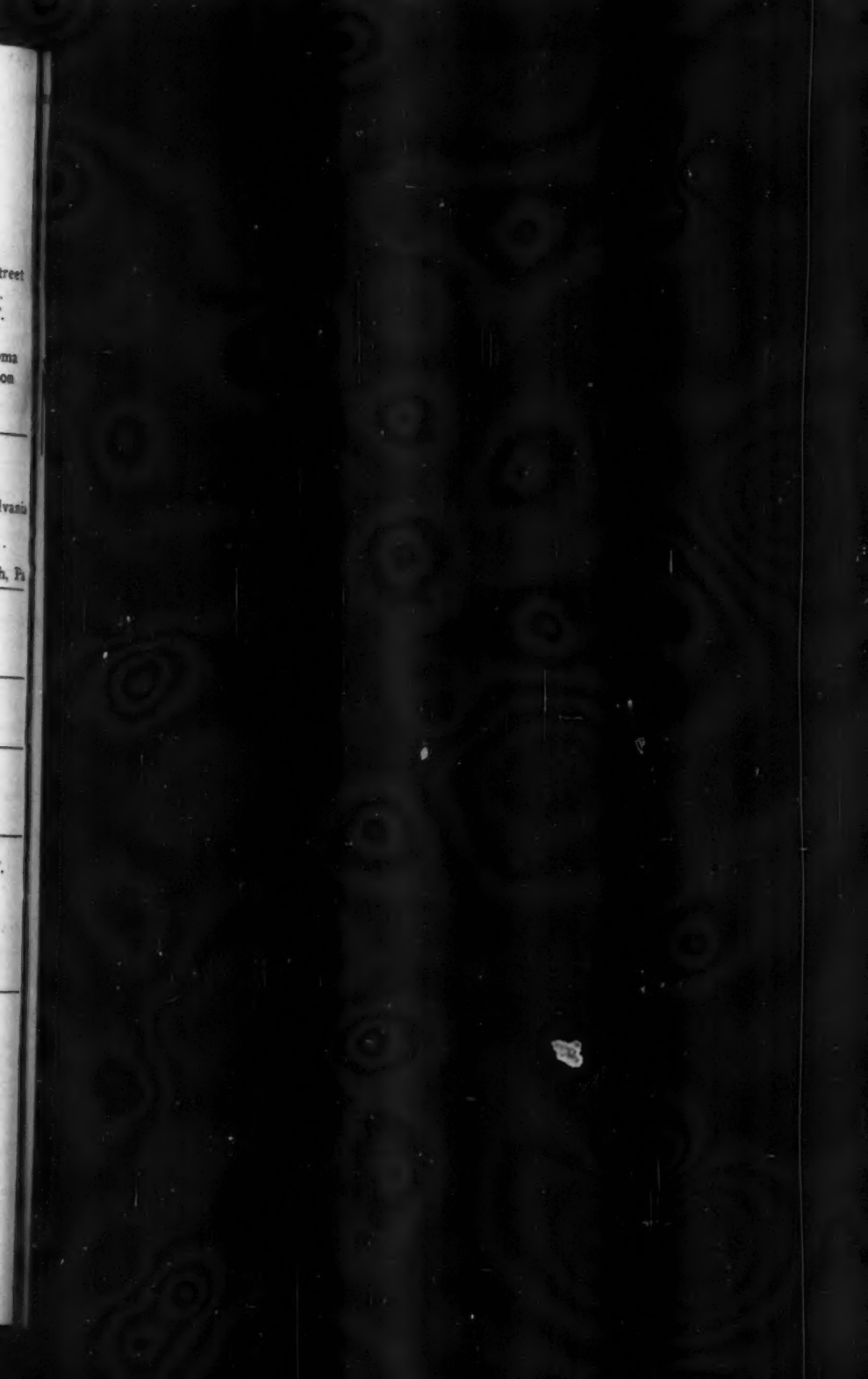
WISCONSIN

Wisconsin Association of M.T. Pres.—Alice A. Thorngate	National	Semi-annual	Mrs. Eliz. Kullman, M.T. 2460 So. 59th Street Milwaukee, Wisconsin
Wisconsin Association of M.T. Milwaukee District Pres.—Dorothy Zoeller	State	Monthly	Esther Lemont, M.T. 2618 North Summit Ave. Milwaukee, Wisconsin

COUNTY SOCIETIES

Travis County Soc. of Medical Technologists (Austin)—Pres., Miss Frances Kelly, c/o Dr. H. J. Gondolf, Austin, Texas.

Tarrant County Society (Fort Worth)—Pres., Miss Ruby L. Oxford, 1805 Alston Ave., Fort Worth 4, Texas.

Harris County Society, Mary Nagai, President, Jefferson Davis Hospital, Houston, Texas.

CECELIA M. KORTUEM, R.N., M.T. (ASCP)
Editorial Staff